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NEWS	2	OCT 04	Precision of EMBASE searching enhanced with new chemical name field
NEWS	3	OCT 06	Increase your retrieval consistency with new formats or for Taiwanese application numbers in CA/CAplus.
NEWS	4	OCT 21	CA/CAplus kind code changes for Chinese patents increase consistency, save time
NEWS	5	OCT 22	New version of STN Viewer preserves custom highlighting of terms when patent documents are saved in .rtf format
NEWS	6	OCT 28	INPADOCDB/INPAFAMDB: Enhancements to the US national patent classification.
NEWS	7	NOV 03	New format for Korean patent application numbers in CA/CAplus increases consistency, saves time.
NEWS	8	NOV 04	Selected STN databases scheduled for removal on December 31, 2010
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NEWS	11	NOV 24	Search an additional 46,850 records with MEDLINE backfile extension to 1946
NEWS	12	DEC 14	New PNK Field Allows More Precise Crossover among STN Patent Databases
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NEWS	14	DEC 21	CAS Learning Solutions -- a new online training experience
NEWS	15	DEC 22	Value-Added Indexing Improves Access to World Traditional Medicine Patents in CAplus
NEWS	16	JAN 24	The new and enhanced DPCI file on STN has been released
NEWS	17	JAN 26	Improved Timeliness of CAS Indexing Adds Value to USPATFULL and USPAT2 Chemistry Patents
NEWS	18	JAN 26	Updated MeSH vocabulary, new structured abstracts, and other enhancements improve searching in STN reload of MEDLINE
NEWS	19	JAN 28	CABA will be updated weekly
NEWS	20	FEB 23	PCTFULL file on STN completely reloaded
NEWS	21	FEB 23	STN AnaVist Test Projects Now Available for Qualified Customers
NEWS	22	FEB 25	LPCI will be replaced by LDPCI
NEWS	23	MAR 07	Pricing for SELECTing Patent, Application, and Priority Numbers in the USPAT and IFI Database Families is Now Consistent with Similar Patent Databases on STN
NEWS	24	APR 26	Expanded Swedish Patent Application Coverage in CA/CAplus Provides More Current and Complete Information
NEWS	25	APR 28	The DWPI (files WPINDEX, WPIDS and WPIX) on STN have been enhanced with thesauri for the European Patent Classifications

NEWS 26 MAY 02 MEDLINE Improvements Provide Fast and Simple Access to DOI and Chemical Name Information
NEWS 27 MAY 12 European Patent Classification thesauri added to the INPADOC files, PCTFULL, GBFULL and FRFULL
NEWS 28 MAY 20 PATDPA database updates to end in June 2011

NEWS EXPRESS 17 DECEMBER 2010 CURRENT WINDOWS VERSION IS V8.4.2 .1,
AND CURRENT DISCOVER FILE IS DATED 24 JANUARY 2011.

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FILE 'HOME' ENTERED AT 11:52:55 ON 20 MAY 2011

FILE 'MERLINE' ENTERED AT 11:53:08 ON 20 MAY 2011

FILE LAST UPDATED: 19 May 2011 (20110519/UP). FILE COVERS 1946 TO DATE.

MEDLINE and LMEDLINE have been updated with the 2011 Medical Subject Headings (MeSH) vocabulary and tree numbers from the U.S. National Library of Medicine (NLM). Additional information is available at:

http://www.nlm.nih.gov/pubs/techbull/nd10/nd10_medline_data_changes_2011.html

The 2011 Medline reload was completed on January 22, 2011.
See [HELP: RLOAD](#) for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

See HELP RANGE before carrying out any RANGE search

→ *s*-catenin and (protein kinase *c* or pks)

12785 CATEIN
1208 CATEINNS
12977 CATEIN
(CATEIN OR CATEINNS)
2210493 PROTEIN
1782640 PROTEINS
2740349 PROTEIN
(PROTEIN OR PROTEINS)
337855 KINASE
199825 KINASES
384940 KINASE
(KINASE OR KINASES)
1389122 C
52343 PROTEIN KINASE C
(PROTEIN(W) KINASE(W) C)
26271 PKC
1623 PKCS
27117 PKC
(PKC OR PKCS)
L1 227 CATEIN AND (PROTEIN KINASE C OR PKC)

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1098 IOTA
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39130 LAMBDA
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L2 39179 L1 AND IOTA OR LAMBDA

=> s 11 and (iota or lambda)
1098 IOTA
1 IOTAS
1099 IOTA
(IOTA OR IOTAS)
39130 LAMBDA
91 LAMBDA
39179 LAMBDA
(LAMBDA OR LAMBDA)
L3 6 L1 AND (IOTA OR LAMBDA)

=> d bib ab 1-6

L3 ANSWER 1 OF 6 MEDLINE on STN
AN 2009319691 MEDLINE
DN PubMed ID: 19290490
TI Expression of P-aPKC-*iota*, E-cadherin, and beta-catenin related to invasion and metastasis in hepatocellular carcinoma.
AU Du Guang-Sheng; Wang Jian-Ming; Lu Jin-Xi; Li Qiang; Ma Chao-Qun; Du Ji-Tao; Zou Sheng-Quan
CS Department of General Surgery, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, Hubei, China.
SO Annals of surgical oncology, (2009 Jun) Vol. 16, No. 6, pp. 1578-86.
Electronic Publication: 2009-03-17.
Journal code: 9420840. E-ISSN: 1534-4681. L-ISSN: 1068-9265.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals

EM 200906

ED Entered STN: 6 May 2009

Last Updated on STN: 19 Jun 2009

Entered Medline: 18 Jun 2009

OSC.G 1 There are 1 MEDLINE records that cite this record

AB OBJECTIVES: Atypical protein kinase C iota (aPKC-iota) and its associated intracellular molecules, E-cadherin and beta-catenin, are important for cell polarization in tumorigenesis and progression. Expression of aPKC-iota, P-aPKC-iota (activated aPKC-iota), E-cadherin, and beta-catenin in hepatocellular carcinoma (HCC) was measured, and correlation with clinicopathological characteristics of HCC was analyzed.

METHODS: Paraffin-embedded tumor tissue was obtained from patients with HCC after resection without preoperative radiotherapy or chemotherapy. Gene expression was detected by polymerase chain reaction (PCR), and protein expression was detected by immunohistochemistry and Western blot analysis. Expressions of aPKC-iota, P-aPKC-iota, E-cadherin, and beta-catenin were analyzed with relation to the clinicopathological data.

RESULTS: The gene and protein expression of aPKC-iota are obviously higher in HCC tissues than that in peritumoral tissues and normal tissues by semiquantitative PCR and immunohistochemistry methods. Accumulation of aPKC-iota in HCC cytoplasm and nucleolus inhibited the later formation of belt-like adherens junctions (AJs) and/or tight junctions (TJs) in cell-cell contact. E-cadherin was reduced and accumulation of cytoplasm beta-catenin was increased in HCC. The expression of aPKC-iota was closely related to pathological differentiation, tumor size, invasion, and metastasis of HCC.

CONCLUSION: Accumulation of cytoplasm aPKC-iota may reflect pathological differentiation, invasion, and metastasis potential of HCC. In this regard, our study on HCC revealed the potential usefulness of aPKC-iota, E-cadherin, and beta-catenin as a prognostic marker, closely related to pathological differentiation, invasion, metastasis, and prognosis of HCC.

L3 ANSWER 2 OF 6 MEDLINE on STN

AN 2009075089 MEDLINE

DN PubMed ID: 19147581

TI Protein kinase C betaII and PKCiota/lambda: collaborating partners in colon cancer promotion and progression.

AU Murray Nicole R; Weems Justin; Braun Ursula; Leitges Michael; Fields Alan P

CS Department of Cancer Biology, Mayo Clinic College of Medicine, Jacksonville, Florida 32224, USA.

NC CA081436 (United States NCI NIH HHS)

CA094122 (United States NCI NIH HHS)

R01 CA081436-11 (United States NCI NIH HHS)

SO Cancer research, (2009 Jan 15) Vol. 69, No. 2, pp. 656-62.
Journal code: 2984705R. E-ISSN: 1538-7445. L-ISSN: 0008-5472.
Report No.: NLM-NIHMS79085; NLM-PMC2688739.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)

LA English

FS Priority Journals

EM 200902

ED Entered STN: 17 Jan 2009

Last Updated on STN: 24 Feb 2009

Entered Medline: 20 Feb 2009

OSC.G 3 There are 3 MEDLINE records that cite this record

REM.CNT 23 There are 23 cited references available in MEDLINE for this document.

AB We previously showed that elevated expression of either protein kinase C β II (PKC β II) or PKC ι /lambda enhances colon carcinogenesis in mice. Here, we use novel bitransgenic mice to determine the relative importance of PKC β II and PKC ι /lambda in colon carcinogenesis in two complimentary models of colon cancer *in vivo*. Bitransgenic mice overexpressing PKC β II and constitutively active PKC ι (PKC β II/caPKC ι) or kinase-deficient, dominant-negative PKC ι (PKC β II/kdPKC ι) in the colon exhibit a similar increase in colon tumor incidence, tumor size, and tumor burden in response to azoxymethane (AOM) when compared with nontransgenic littermates. However, PKC β II/kdPKC ι mice develop predominantly benign colonic adenomas, whereas PKC β II/caPKC ι mice develop malignant carcinomas. In contrast, PKC β -deficient (PKC β (-/-)) mice fail to develop tumors even in the presence of caPKC ι . Our previous data indicated that PKC β II drives tumorigenesis and proliferation by activating beta-catenin/Apc signaling. Consistent with this conclusion, genetic deletion of PKC β has no effect on spontaneous tumorigenesis in Apc(min $+$) mice. In contrast, tissue-specific knockout of PKC λ significantly suppresses intestinal tumor formation in Apc(min $+$) mice. Our data show that PKC β II and PKC ι /lambda serve distinct, nonoverlapping functions in colon carcinogenesis. PKC β II is required for AOM-induced tumorigenesis but is dispensable for tumor formation in Apc(Min $+$) mice. PKC ι /lambda promotes tumor progression in both AOM- and Apc(min $+$)-induced tumorigenesis. Thus, PKC β II and PKC ι , whose expression is elevated in both rodent and human colon tumors, collaborate to drive colon tumor formation and progression, respectively.

L3 ANSWER 3 OF 6 MEDLINE on STN
AN 2008503980 MEDLINE
DN PubMed ID: 18466329
TI Regulation of neural progenitor cell motility by ceramide and potential implications for mouse brain development.
AU Wang Guanghu; Krishnamurthy Kannan; Chiang Ying-Wei; Dasgupta Somsankar; Bieberich Erhard
CS Program in Developmental Neurobiology, Institute of Molecular Medicine and Genetics, School of Medicine, Medical College of Georgia, Augusta, Georgia, USA.
NC R01NS046835 (United States NINDS NIH HHS)
SO Journal of neurochemistry, (2008 Jul) Vol. 106, No. 2, pp. 718-33.
Electronic Publication: 2008-05-02.
Journal code: 2985190R. E-ISSN: 1471-4159. L-ISSN: 0022-3042.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
LA English
FS Priority Journals
EM 200809
ED Entered STN: 9 Aug 2008
Last Updated on STN: 17 Sep 2008
Entered Medline: 16 Sep 2008
OSC.G 1 There are 1 MEDLINE records that cite this record
AB We provide evidence that the sphingolipid ceramide, in addition to its pro-apoptotic function, regulates neural progenitor (NP) motility *in vitro* and brain development *in vivo*. Ceramide (N-palmitoyl d-erythro sphingosine and N-oleoyl d-erythro sphingosine) and the ceramide analog N-oleoyl serinol (S18) stimulate migration of NPs in scratch (wounding) migration assays. Sphingolipid depletion by inhibition of de novo ceramide biosynthesis, or ceramide inactivation using an anti-ceramide antibody, obliterates NP motility, which is restored by ceramide or S18. These results suggest that ceramide is crucial for NP motility. Wounding

of the NP monolayer activates neutral sphingomyelinase indicating that ceramide is generated from sphingomyelin. In membrane processes, ceramide is co-distributed with its binding partner atypical protein kinase C zeta/lambda (aPKC), and Cdc42, alpha/beta-tubulin, and beta-catenin, three proteins involved in aPKC-dependent regulation of cell polarity and motility. Sphingolipid depletion by myriocin prevents membrane translocation of aPKC and Cdc42, which is restored by ceramide or S18. These results suggest that ceramide-mediated membrane association of aPKC/Cdc42 is important for NP motility. In vivo, sphingolipid depletion leads to ectopic localization of mitotic or post-mitotic neural cells in the embryonic brain, while S18 restores the normal brain organization. In summary, our study provides novel evidence that ceramide is critical for NP motility and polarity in vitro and in vivo.

L3 ANSWER 4 OF 6 MEDLINE on STN
AN 2006668260 MEDLINE
DN PubMed ID: 16959241
TI Beta-catenin is essential for lamination but not neurogenesis in mouse retinal development.
AU Fu Xueyao; Sun Hongxia; Klein William H; Mu Xiuqian
CS Department of Biochemistry and Molecular Biology, Unit 1000, The University of Texas M. D. Anderson Cancer Center, 1515 Holcombe Blvd., Houston, TX 77030, USA.
NC CA16672 (United States NCI NIH HHS)
EY011930 (United States NEI NIH HHS)
R01 EY011930-07 (United States NEI NIH HHS)
R01 EY011930-08 (United States NEI NIH HHS)
R01 EY011930-09 (United States NEI NIH HHS)
SO Developmental biology, (2006 Nov 15) Vol. 299, No. 2, pp. 424-37.
Electronic Publication: 2006-08-10.
Journal code: 0372762. ISSN: 0012-1606. L-ISSN: 0012-1606.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200701
ED Entered STN: 21 Nov 2006
Last Updated on STN: 17 Jan 2007
Entered Medline: 16 Jan 2007
OSC.G 12 There are 12 MEDLINE records that cite this record
AB During vertebrate retinal development, the seven retinal cell types differentiate sequentially from a single population of retinal progenitor cells (RPCs) and organize themselves into a distinct laminar structure. The purpose of this study was to determine whether beta-catenin, which functions both as a nuclear effector for the canonical Wnt signaling pathway and as a regulator of cell adhesion, is required for retinal neurogenesis or lamination. We used the Cre-loxP system to either eliminate beta-catenin or to express a constitutively active form during retinal neurogenesis. Eliminating beta-catenin did not affect cell differentiation, but did result in the loss of the radial arrangement of RPCs and caused abnormal migration of differentiated neurons. As a result, the laminar structure was massively disrupted in beta-catenin-null retinas, although all retinal cell types still formed. In contrast to other neural tissues, eliminating beta-catenin did not significantly reduce the proliferation rate of RPCs; likewise, activating beta-catenin ectopically in RPCs did not result in overproliferation, but loss of neural retinal identity. These results indicate that beta-catenin is essential during retinal neurogenesis as a regulator of cell adhesion but not as a nuclear effector of the canonical Wnt signaling pathway. The results further imply that retinal lamination and retinal

cell differentiation are genetically separable processes.

L3 ANSWER 5 OF 6 MEDLINE on STN
AN 2006345827 MEDLINE
DN PubMed ID: 16478782
TI Regulation of dishevelled and beta-catenin in rat skeletal muscle: an alternative exercise-induced GSK-3beta signaling pathway.
AU Aschenbach William G; Ho Richard C; Sakamoto Kei; Fujii Nobuharu; Li Yangfeng; Kim Young-Bum; Hirshman Michael F; Goodyear Laurie J
CS Joslin Diabetes Center, One Joslin Place, Boston, MA 02215, USA.
NC F32-AR-049662 (United States NIAMS NIH HHS)
F32-DK-59769 (United States NIDDK NIH HHS)
R01-AR-42238 (United States NIAMS NIH HHS)
R01-DK-68626 (United States NIDDK NIH HHS)
SO American journal of physiology. Endocrinology and metabolism, (2006 Jul)
Vol. 291, No. 1, pp. E152-8. Electronic Publication: 2006-02-14.
Journal code: 100901226. ISSN: 0193-1849. L-ISSN: 0193-1849.
CY United States
DT (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
LA English
FS Priority Journals
EM 200608
ED Entered STN: 9 Jun 2006
Last Updated on STN: 4 Aug 2006
Entered Medline: 3 Aug 2006
OSC.G 2 There are 2 MEDLINE records that cite this record
AB beta-catenin is a multifunctional protein involved in cell-cell adhesion and the Wnt signaling pathway. beta-Catenin is activated upon its dephosphorylation, an event triggered by Dishevelled (Dvl)-mediated phosphorylation and deactivation of glycogen synthase kinase-3beta (GSK-3beta). In skeletal muscle, both insulin and exercise decrease GSK-3beta activity, and we tested the hypothesis that these two stimuli regulate beta-catenin. Immunoblotting demonstrated that Dvl, Axin, GSK-3beta, and beta-catenin proteins are expressed in rat red and white gastrocnemius muscles. Treadmill running exercise *in vivo* significantly decreased beta-catenin phosphorylation in both muscle types, with complete dephosphorylation being elicited by maximal exercise. beta-Catenin dephosphorylation was intensity dependent, as dephosphorylation was highly correlated with muscle glycogen depletion during exercise ($r^2 = 0.84$, $P < 0.001$). beta-Catenin dephosphorylation was accompanied by increases in GSK-3beta Ser(9) phosphorylation and Dvl-GSK-3beta association. In contrast to exercise, maximal insulin treatment (1 U/kg body wt) had no effect on skeletal muscle beta-catenin phosphorylation or Dvl-GSK-3beta interaction. In conclusion, exercise *in vivo*, but not insulin, increases the association between Dvl and GSK-3beta in skeletal muscle, an event paralleled by beta-catenin dephosphorylation.

L3 ANSWER 6 OF 6 MEDLINE on STN
AN 2005255330 MEDLINE
DN PubMed ID: 15665047
TI Potential candidates for ischemic preconditioning-associated vascular growth pathways revealed by antibody array.
AU Mathur Praveer; Kaga Shigeaki; Zhan Lijun; Das Dipak K; Maulik Nilanjana
CS Molecular Cardiology Laboratory, Dept. of Surgery, Univ. of Connecticut School of Medicine, Farmington, CT 06030-1110, USA.
NC HL-22559 (United States NHLBI NIH HHS)
HL-33889 (United States NHLBI NIH HHS)
HL-56803 (United States NHLBI NIH HHS)
HL-69910 (United States NHLBI NIH HHS)
SO American journal of physiology. Heart and circulatory physiology, (2005

Jun) Vol. 288, No. 6, pp. H3006-10. Electronic Publication: 2005-01-21.
Journal code: 100901228. ISSN: 0363-6135. L-ISSN: 0363-6135.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LA English
FS Priority Journals
EM 200507
ED Entered STN: 18 May 2005
Last Updated on STN: 8 Jul 2005
Entered Medline: 7 Jul 2005

OSC.G 3 There are 3 MEDLINE records that cite this record

AB Our understanding of the phenomenon of myocardial vascular growth is very limited even though various studies have been conducted in several different models, because the focus in each has been on a select very few number of proteins as the possible growth factors. In the present study, we used the ischemic preconditioning (IP) model in the form of four in vivo repetitive cycles of coronary artery occlusion, each followed by reperfusion as the model to stimulate vascular growth, and performed the protein profiling using high-throughput antibody array technology. Rats were divided into two groups: control + left anterior descending coronary artery (LAD) occlusion (CMI), and IP+ LAD occlusion (IPMI). The antibody array experiment performed to compare the expression of 512 proteins between the IPMI and CMI samples revealed significant upregulation of growth proteins like TGF-beta, BMX, granulocyte-monocyte colony-stimulating factor, signal transducer and activator of transcription 3, alpha- and beta-catenins, ubiquitin-conjugating enzyme UbcH6, nexilin, and PKC-epsilon and -lambda. JNK1 and c-Src tyrosine kinase were expectedly found to be downregulated. Western blot experiments validated the changes in expression of these proteins. Therefore, this study puts forward the above-mentioned proteins as valid participants in the vascular growth signals that are known to be triggered by ischemic preconditioning of heart.

=> d his full

(FILE 'HOME' ENTERED AT 11:52:55 ON 20 MAY 2011)

FILE 'MEDLINE' ENTERED AT 11:53:08 ON 20 MAY 2011
L1 227 SEA PLU=ON CATEIN AND (PROTEIN KINASE C OR PKC)
L2 39179 SEA PLU=ON L1 AND IOTA OR LAMBDA
L3 6 SEA PLU=ON L1 AND (IOTA OR LAMBDA)
D BIB AB 1-6

FILE HOME

FILE MEDLINE

FILE LAST UPDATED: 19 May 2011 (20110519/UP). FILE COVERS 1946 TO DATE.

MEDLINE and LMEDLINE have been updated with the 2011 Medical Subject Headings (MeSH) vocabulary and tree numbers from the U.S. National Library of Medicine (NLM). Additional information is available at:

http://www.nlm.nih.gov/pubs/techbull/nd10/nd10_medline_data_changes_2011.

The 2011 Medline reload was completed on January 22, 2011.
See HELP RLOAD for details.

This file contains CAS Registry Numbers for easy and accurate

substance identification.

See HELP RANGE before carrying out any RANGE search.

```
=> s catenin and apkc and iota
    12785 CATENIN
    1208 CATENINS
    12977 CATENIN
        (CATENIN OR CATENINS)
    414 APKC
    60 APKCS
    431 APKC
        (APKC OR APKCS)
1098 IOTA
    1 IOTAS
1099 IOTA
        (IOTA OR IOTAS)
L4      1 CATEININ AND APKC AND IOTA
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=> s l4 not l3
L5      0 L4 NOT L3
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(FILE 'HOME' ENTERED AT 11:52:55 ON 20 MAY 2011)

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FILE 'MEDLINE' ENTERED AT 11:53:08 ON 20 MAY 2011
L1      227 SEA PLU=ON CATEININ AND (PROTEIN KINASE C OR PKC)
L2      39179 SEA PLU=ON L1 AND IOTA OR LAMBDA
L3      6 SEA PLU=ON L1 AND (IOTA OR LAMBDA)
        D BIB AB 1-6
L4      1 SEA PLU=ON CATEININ AND APKC AND IOTA
L5      0 SEA PLU=ON L4 NOT L3
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FILE HOME

FILE MEDLINE

FILE LAST UPDATED: 19 May 2011 (20110519/UP). FILE COVERS 1946 TO DATE.

MEDLINE and LMEDLINE have been updated with the 2011 Medical Subject Headings (MeSH) vocabulary and tree numbers from the U.S. National Library of Medicine (NLM). Additional information is available at:

http://www.nlm.nih.gov/pubs/techbull/nd10/nd10_medline_data_changes_2011.

The 2011 Medline reload was completed on January 22, 2011.
See HELP RLOAD for details.

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See HELP RANGE before carrying out any RANGE search.

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COST IN U.S. DOLLARS          SINCE FILE      TOTAL
                                ENTRY          SESSION
FULL ESTIMATED COST          5.34          5.57
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STN INTERNATIONAL LOGOFF AT 11:58:35 ON 20 MAY 2011